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=> file medline

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0.63 0.63

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MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2005 vocabulary.

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=> s (inhibit delay) (P) (cytokinesis cytodieresis interphase) and (chemotherapeutic anticancer antineoplastic) and (cytochalasin dihydroxytochalasin jasplakinolide chondramide latrunculin)

138193 INHIBIT

63618 DELAY

12 INHIBIT DELAY

(INHIBIT (W) DELAY)

3488 CYTOKINESIS

10 CYTODIERESIS

12634 INTERPHASE

O CYTOKINESIS CYTODIERESIS INTERPHASE

(CYTOKINESIS (W) CYTODIERESIS (W) INTERPHASE)

0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE)

19947 CHEMOTHERAPEUTIC

17245 ANTICANCER

187866 ANTINEOPLASTIC

O CHEMOTHERAPEUTIC ANTICANCER ANTINEOPLASTIC

(CHEMOTHERAPEUTIC (W) ANTICANCER (W) ANTINEOPLASTIC)

9841 CYTOCHALASIN

0 DIHYDROXYTOCHALASIN

240 JASPLAKINOLIDE

6 CHONDRAMIDE

602 LATRUNCULIN

O CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE LATRUNCULIN

(CYTOCHALASIN (W) DIHYDROXYTOCHALASIN (W) JASPLAKINOLIDE (W) CHONDRA MIDE (W) LATRUNCULIN)

0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE) AND (CHEMOTHERAPEUTIC ANTICANCER ANTINEOPLASTIC) AND (CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE LATRUNCULIN)

=> s (inhibit delay) (P) (cytokinesis cytodieresis interphase) and (cytochalasin dihydroxytochalasin jasplakinolide chondramide latrunculin)

T.1

138193 INHIBIT

63618 DELAY

12 INHIBIT DELAY

(INHIBIT (W) DELAY)

3488 CYTOKINESIS

10 CYTODIERESIS

12634 INTERPHASE

O CYTOKINESIS CYTODIERESIS INTERPHASE

(CYTOKINESIS (W) CYTODIERESIS (W) INTERPHASE)

O (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE)

9841 CYTOCHALASIN

0 DIHYDROXYTOCHALASIN

240 JASPLAKINOLIDE

6 CHONDRAMIDE

602 LATRUNCULIN

O CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE LATRUNCULIN

(CYTOCHALASIN(W)DIHYDROXYTOCHALASIN(W)JASPLAKINOLIDE(W)CHONDRA MIDE(W)LATRUNCULIN)

0 (INHIBIT DELAY) (P) (CYTOKINESIS CYTODIERESIS INTERPHASE) AND (CYTOCHALASIN DIHYDROXYTOCHALASIN JASPLAKINOLIDE CHONDRAMIDE LATRUNCULIN)

=> s (inhibit delay) (P) (cytokinesis cytodieresis interphase) and "cytochalasin dihydroxytochalasin jasplakinolide chondramide latrunculin)
MISMATCHED QUOTE 'AND "CYTOCHALAS'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s inhibit or delay

L2

138193 INHIBIT

63618 DELAY

L3 200566 INHIBIT OR DELAY

=> 13 (P) cytokinesis

L3 IS NOT A RECOGNIZED COMMAND

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=> 13(P)cytokinesis

L3 (P) CYTOKINESIS IS NOT A RECOGNIZED COMMAND

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=> 13 and cytokenesis

L3 IS NOT A RECOGNIZED COMMAND

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=> s 13 (P) cytokinesis

3488 CYTOKINESIS

206 L3 (P) CYTOKINESIS

=> s 14 and cytochalasin

9841 CYTOCHALASIN

23 L4 AND CYTOCHALASIN

=> d 20-23 bib abs

L4

L5

L5 ANSWER 20 OF 23 MEDLINE on STN

AN 81168410 MEDLINE

- PubMed ID: 6111566 DN
- A permeabilized cell model for studying cell division: a comparison of TI anaphase chromosome movement and cleavage furrow constriction in lysed PtK1 cells.
- Cande W Z; McDonald K; Meeusen R L ΑU
- BRSI 507 RR0706 (NCRR) NC GM 23238 (NIGMS)
- Journal of cell biology, (1981 Mar) 88 (3) 618-29. SO Journal code: 0375356. ISSN: 0021-9525.
- CY United States
- Journal; Article; (JOURNAL ARTICLE) DT
- LΑ English
- FS Priority Journals
- EM 198106
- ED Entered STN: 19900316

Last Updated on STN: 19970203

Entered Medline: 19810613

After lysis in a Brij 58-polyethylene glycol medium, PtK1 cells are AΒ permeable to small molecules, such as erythrosin B, and to proteins, such as rhodamine-labeled FAB, myosin subfragment-1, and tubulin. Holes are present in the plasma membrane, and the mitochondria are swollen and distorted, but other membrane-bounded organelles of the lysed cell model are not noticeably altered. After lysis, the mitotic apparatus is functional; chromosomes move poleward and the spindle elongates. Cells lysed while in cytokinesis will continue to divide for several minutes. Addition of crude tubulin extracts, MAP-free tubulin, or taxol to the lysis medium retards anaphase chromosome movements but does not affect cleavage. On the other hand, N-ethylmaleimide-modified myosin subfragment-1, phalloidin, and cytochalasin B inhibit cleavage but have no effect on anaphase chromosome movements under identical lysis conditions. These results suggest that actomyosin plays no functional role in anaphase chromosome movement in mammalian tissue culture cells and that microtubule depolymerization is a rate-limiting step for chromosome-to-pole movements.

- ANSWER 21 OF 23 MEDLINE on STN L5
- AN 80134838 MEDLINE
- DN PubMed ID: 6444598
- TI · Ultrastructural changes in the embryonic cells of the frog Microhyla ornata after cytochalasin H treatment.
- Wadekar G; Dastur D K; Mulherkar L ΑU
- Experimental cell biology, (1980) 48 (2) 155-66. SO Journal code: 7701827. ISSN: 0304-3568.
- CY Switzerland
- Journal; Article; (JOURNAL ARTICLE) DT
- LA English
- Priority Journals FS
- EM198005
- ED Entered STN: 19900315

Last Updated on STN: 19900315

Entered Medline: 19800523

Biological effects of cytochalasin H (CH), a newly isolated AB mould metabolite, have been found to bring about disaggregation of embryonic cells and to inhibit cytokinesis.

Disaggregation is known to be a phenomenon related to the cell surface. (The cells are held together by a mucopolysaccharide glycoprotein complex.) In the present work the fact that the mucopolysaccharide glycoprotein surface coat gets affected by CH treatment is confirmed by electron microscopy with the help of Lanthanum, a specific marker, which gets selectively absorbed to the cell surface material and renders it electron dense. The ultrastructural observations indicated the reduction of the cell surface material in treated embryos as compared to the controls. The reappearance of lanthanum-bound cell surface material in the recovered embryos was also observed. However, the exact mechanism of the action of CH on the cell surface remains to be clarified.

- L5 ANSWER 22 OF 23 MEDLINE on STN AN 77235913 MEDLINE
- DN PubMed ID: 882839
- TI Cytochalasin B partially inhibits the oxalate-induced radial segmentation of mononucleated blood cells.
- AU Simmingskold G; Rydgren L; Norberg B; Soderstrom U B; Ponten J
- SO Scandinavian journal of haematology, (1977 Jul) 19 (1) 33-8. Journal code: 0404507. ISSN: 0036-553X.
- CY Denmark
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 197709
- ED Entered STN: 19900314 Last Updated on STN: 19900314 Entered Medline: 19770917
- AB Cytochalasin B (CB), 5 microgram/ml (= 1.0 x 10(-5) M), inhibited the oxalate-induced radial segmentation of the nuclei of lymphocytes and monocytes from peripheral blood. The median inhibition was 60%. The oxalate-induced radial segmentation (RS) is thought to be due to a microtubule-dependent contraction of the intermitotic residue of the mitotic apparatus around the nucleus. CB is thought to inhibit cell locomotion and cytokinesis by a centripetal contraction of the membrane-associated contractile cell layer without subsequent relaxation. It is thus suggested that the CB inhibition of the oxalate-induced RS was due to a spatial interference of the CB-induced contraction with the formation of RS nuclei.
- L5 ANSWER 23 OF 23 MEDLINE on STN
- AN 76027739 MEDLINE
- DN PubMed ID: 1165581
- TI Membrane effects of cytochalasin B. Competitive inhibition of facilitated diffusion processes in rat hepatoma cells and other cell lines and effect on formation of functional transport sites.
- AU Plagemann P G; Zylka J H; Erbe J; Estensen R D
- SO Journal of membrane biology, (1975 Aug 11) 23 (1) 77-90. Journal code: 0211301. ISSN: 0022-2631.
- CY United States
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 197512
- ED Entered STN: 19900313 Last Updated on STN: 19970203 Entered Medline: 19751212
- Cytochalasin B competitively inhibits the transport of AB 2-deoxy-D-glucose and thymidine in a number of different cell lines (Novikoff rat hepatoma cells, mouse L, S180 and Ki-MSV-transformed BALB/3T3 cells, and human HeLa cells). The apparent Km values for the transport of these substrates as well as the apparent Ki values for the inhibition by cytochalasin B are very similar for the various cell lines, and the effect is readily and completely reversed by removal of the chemical. Thymidine transport by Chinese hamster ovary cells however, is little affected by cytochalasin B, whereas the transport of 2-deoxy-D-glucose, uridine and guanine by these cells is competitively inhibited to about the same extent as in other cell lines. In addition and concomitant with the inhibition of cytokinesis and an alteration in cell shape, cytochalasin B also impairs and delays the formation of functional transport sites for thymidine, guanine and choline in synchronized populations of Novikoff cells without affecting the apparent affinities of the transport systems for their substrates. This effect is unrelated to the direct inhibition of the transport processes, since the drug does not directly inhibit choline transport and has no effect on the formation of 2-deoxy-D-glucose

transport sites in spite of the fact that it strongly inhibits the transport of this substrate. The inhibition of functional transport sites may be due to the induction of a structural alteration in the membrane by cytochalasin B which impairs the insertion of new proteins of certain but not all transport systems into the membrane.

=> s 14 and cytochalasin and chemotherapeutic 9841 CYTOCHALASIN 19947 CHEMOTHERAPEUTIC 1 L4 AND CYTOCHALASIN AND CHEMOTHERAPEUTIC

=> d 1 abs bib

ANSWER 1 OF 1 MEDLINE on STN L6

INTRODUCTION: The in vitro chemosensitivity testing aims at predicting the AB response of an individual tumour to chemotherapy choosing optimal agents for a particular patient. Among many chemosensitivity tests developed over the years [1-6], special emphasis was made on clonogenic assays that showed good use and correlation between laboratory and clinical data [7-9]. One of the assays used to predict the response to various anti-cancer modalities is the micronucleus assay using the cytokinesis-block [12-14]. This block is achieved by administration of Cytochalasin-B in order to prevent cytoplasmic, but not the nuclear, division. This leads to micronucleus formation which are counted in binuclear cells. Since there are only a few reports of the use of this assay in predicting chemosensitivity [13, 16], we explored the possibility of using this assay to predict chemosensitivity to various anti-cancer agents. MATERIAL AND METHODS: Exponentially growing SCC VII cells were treated with various concentrations of 11 anti-cancer agents: Mitomycin C, Doxorubicin (ADR), Epirubicin (EPI), Cisplatin, Carboplatin (CBDCA), Etoposide (VP-16), Vincristine, 5-fluorouracil, Methotrexate, Nimustine, and Dacarbazine for 1 hour. After that, Cytochalasin-B was added and dishes were incubated. After various time intervals, cells were fixed in situ and dried. Electron microscope was used to count the number of micronuclei (MN) in binucleate cells as well as multinucleate cells (MNC) in the total cell population. Cell survival was also evaluated by using the colony formation assay [18]. RESULTS: Maximal % of binucleate cells (BNC) was usually reached at 24-30 hours of culture, except for cells treated with ADR and EPI, in which it was reached at 30-72 hours (Figures 1 and 2). All drugs induced formation of micronuclei and dose-response curves for micronucleus frequency were obtained using the data at peak % BNC times. For all drugs, micronucleus frequency increased with concentration (Figure 3), but at the highest concentration used (considered to be overly toxic-Figure 4), the micronucleus frequency was rather lower. This decrease in micronucleus frequency was largely attributed to the decrease in % BNC. When the data at the highest concentrations of all drugs were excluded, a correlation was found between micronucleus frequency and surviving fraction (r = 0.85; p < 0.001) (Figure 5). DISCUSSION: Since micronucleus formation is a sign of chromosome damage that leads to cell death, we used this assay to evaluate chemosensitivity in 11 widely used anticancer agents. Although they can be classified according to mechanism of action as different class agents, they have in common the formation of micronuclei as a sign of cytotoxicity. Cell cycle arrest observed in some agents might be evaluated by assessing the delay in increase of BNC and MNC. The difference observed regarding cell cycle arrest suggested different mechanisms of its action. MN frequency was almost dose-dependent at lower concentrations, but at the highest concentrations, it obviously decreased, showing, therefore, some discrepancies with the data obtained when radiosensitivity was tested that way [14], probably due to extreme toxicity of agents. The optimal concentrations seem to be those providing a 20-80% surviving fraction. Another slight difference, when compared with similar radiosensitivity studies is a decrease with longer duration of culture observed in chemosensitivity testings.

reason for this difference is still unknown, but it emphasized the necessity for choosing the optimal duration of culture, probably that necessary for reaching maximal % BNC. This assay seems useful in predicting chemosensitivity of at least some tumour cells to various (appropriate) concentrations of various anti-cancer agents. However, new studies are warranted to further use of this assay, before testing it in clinical practice.

AN 97206774 MEDLINE

DN PubMed ID: 9102841

TI [Significance of formation of micronuclei in SCC VII murine cells treated with various chemotherapeutic agents].

Znacaj formiranja mikronukleusa u murinim celijama SCC VII tretiranih razlicitim hemioterapijskim agensima.

AU Jeremic B; Sibamoto J; Abe M

CS Department of Oncology, Clinical-Hospital Centre, Kragujevac, Yugoslavia.

SO Srpski arhiv za celokupno lekarstvo, (1996 Jul-Aug) 124 (7-8) 169-74. Journal code: 0027440. ISSN: 0370-8179.

CY Yugoslavia

DT Journal; Article; (JOURNAL ARTICLE)

LA Serbian

FS Priority Journals

EM 199704

ED Entered STN: 19970424

Last Updated on STN: 19970424 Entered Medline: 19970416

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FILE LAST UPDATED: 29 JUN 2005 (20050629/UP). FILE COVERS 1950 TO DATE.

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26 FILES SEARCHED...
    10508
            FILE DGENE
     920
            FILE DISSABS
    21018
            FILE DRUGB
            FILE DRUGMONOG2
    1376
            FILE DRUGU
    60653
            FILE EMBAL
      604
            FILE EMBASE
  142055
   15970
            FILE ESBIOBASE
    1649
            FILE FEDRIP.
            FILE FROSTI
       27
       19.
            FILE FSTA
     1321
            FILE GENBANK
     148
            FILE HEALSAFE
    3830
            FILE IFIPAT
            FILE IMSDRUGNEWS
     1065
            FILE IMSPRODUCT
     983
            FILE IMSRESEARCH
     468
     5037
            FILE JICST-EPLUS
       25
            FILE KOSMET
     5610
            FILE LIFESCI
    80170
            FILE MEDLINE
      468
            FILE NIOSHTIC
      274
            FILE NTIS
       11
            FILE NUTRACEUT
       19
            FILE OCEAN
    30273
            FILE PASCAL
     1109
            FILE PHAR
     1231
            FILE PHARMAML
57 FILES SEARCHED...
       19
            FILE PHIC
     2764
            FILE PHIN
     7067
            FILE PROMT
     1088
            FILE PROUSDDR
       10
            FILE PS
       5
            FILE RDISCLOSURE
    52266
            FILE SCISEARCH
       50
            FILE SYNTHLINE
   110850
            FILE TOXCENTER
    30192
            FILE USPATFULL
```

```
2297 FILE USPAT2
153 FILE VETB
507 FILE VETU
16 FILE WATER
3822 FILE WPIDS
30 FILE WPIFV
3822 FILE WPINDEX
```

- 71 FILES HAVE ONE OR MORE ANSWERS, 74 FILES SEARCHED IN STNINDEX
- L1 QUE CYTARABINE OR THIOGUANINE OR FLUDARABINE OR FLOXURIDINE OR METHOTREXAT E OR VINCRISTINE OR VINBLASTINE OR PACLITAXEL OR DOCETAXEL OR IRINOTEC AN

| => d | rank | |
|------------|--------|---------------------|
| F1 | 142055 | EMBASE |
| F2 | 110850 | TOXCENTER |
| F3 | 80170 | MEDLINE |
| F4 | 70329 | BIOSIS |
| F5 | 63374 | CANCERLIT |
| F6 | 60653 | DRUGU |
| F7 | 58423 | DDFU |
| F8 | 52266 | SCISEARCH |
| F9 | 37382 | CAPLUS |
| F10 | 30273 | PASCAL |
| F11 | 30192 | USPATFULL |
| F12 | 22232 | ADISCTI |
| F13 | 21018 | DDFB |
| F14 | 21018 | DRUGB |
| F15 | 20038 | BIOTECHNO |
| F16 | 15970 | ESBIOBASE |
| F17 | 10508 | DGENE |
| F18 | 7067 | PROMT |
| F19 | 5610 | LIFESCI |
| F20 | 5037 | JICST-EPLUS |
| F21 | 3830 | IFIPAT |
| F22 | 3822 | WPIDS |
| F23 | 3822 | WPINDEX |
| F24 | 3507 | ADISNEWS |
| F25 | 2764 | PHIN |
| F26 | 2355 | CONFSCI |
| F27 | 2333 | USPAT2 |
| F28 | 2132 | CABA |
| F29 | 1649 | FEDRIP |
| F30 | 1376 | DRUGMONOG2 |
| | 1349 | BIOTECHABS |
| F31
F32 | | BIOTECHDS |
| | 1349 | |
| F33 | 1321 | GENBANK
PHARMAML |
| F34 | 1231 | |
| F35
F36 | 1230 | CIN |
| | 1109 | PHAR |
| F37 | 1088 | PROUSDDR |
| F38 | 1065 | IMSDRUGNEWS |
| F39 | 983 | IMSPRODUCT |
| F40 | 921 | BIOBUSINESS |
| F41 | 920 | DISSABS |
| F42 | 745 | BIOENG |
| F43 | 660 | ADISINSIGHT |
| F44 | 614 | ANABSTR |
| F45 | 604 | EMBAL |
| F46 | 518 | AGRICOLA |
| F47 | 507 | VETU |
| F48 | 468 | IMSRESEARCH |
| F49 | 468 | NIOSHTIC |
| F50 | 274 | NTIS |

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F51
           173
                 BIOCOMMERCE
F52
           160
                 CEABA-VTB
           153
F53
                 VETB
F54
           148
                 HEALSAFE
F55
           107
                 AQUASCI
F56
            69
                 CEN
F57
            50
                 SYNTHLINE
F58
            40
                 CROPU
F59
            30
                 WPIFV
F60
            27
                 FROSTI
F61
            25
                 KOSMET
F62
            24
                 ANTE
F63
            23
                 CROPB
F64
            19
                 FSTA
F65
            19
                 OCEAN
            19
                 PHIC
F66
            16
                 WATER
F67
F68
            12
                 AQUALINE
            11
                 NUTRACEUT
F69
            10
                 PS
F70
                 RDISCLOSURE
             5
F71
```

=> file toxcenter and medline

'AND' IS NOT A VALID FILE NAME

Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.

ENTER A FILE NAME OR (IGNORE): ENTER A FILE NAME OR (IGNORE):end

=> file medline

COST IN U.S. DOLLARS

SINCE FILE TOTAL ENTRY SESSION 2.36 2.57

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 18:39:14 ON 29 JUN 2005

FILE LAST UPDATED: 29 JUN 2005 (20050629/UP). FILE COVERS 1950 TO DATE.

On December 19, 2004, the 2005 MeSH terms were loaded.

The MEDLINE reload for 2005 is now available. For details enter HELP RLOAD at an arrow promt (=>). See also:

http://www.nlm.nih.gov/mesh/

http://www.nlm.nih.gov/pubs/techbull/nd04/nd04 mesh.hcml

OLDMEDLINE now back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2005 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s cytarabine or thioguanine or fludarabine or floxuridine or methotrexate or vincristine or vinblastine or paclitaxel or docetaxel or irinotecan

11453 CYTARABINE

3239 THIOGUANINE

1969 FLUDARABINE

2853 FLOXURIDINE

29781 METHOTREXATE

19432 VINCRISTINE

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2979 DOCETAXEL
         2544 IRINOTECAN
L2
         80170 CYTARABINE OR THIOGUANINE OR FLUDARABINE OR FLOXURIDINE OR METHO
               TREXATE OR VINCRISTINE OR VINBLASTINE OR PACLITAXEL OR DOCETAXEL
                OR IRINOTECAN
=> s 12 and (cytochalasin or dihyrocytochalasin or jasplakinolide or chondramide or
latrunculin)
          9843 CYTOCHALASIN
             0 DIHYROCYTOCHALASIN
           240 JASPLAKINOLIDE
             6 CHONDRAMIDE
           602 LATRUNCULIN
           669 L2 AND (CYTOCHALASIN OR DIHYROCYTOCHALASIN OR JASPLAKINOLIDE OR
L3
               CHONDRAMIDE OR LATRUNCULIN)
=> s 13 and cancer and (interphase or cytodieresis or cytokineses)
        500869 CANCER
         12639 INTERPHASE
            10 CYTODIERESIS
            14 CYTOKINESES
             O L3 AND CANCER AND (INTERPHASE OR CYTODIERESIS OR CYTOKINESES)
L4
=> s 13 and cancer and (interphase or cytodieresis or cytokinesis)
        500869 CANCER
         12639 INTERPHASE
            10 CYTODIERESIS
          3491 CYTOKINESIS
             2 L3 AND CANCER AND (INTERPHASE OR CYTODIERESIS OR CYTOKINESIS)
L5
=> d 1-2 bib asb
'ASB' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'
The following are valid formats:
The default display format is BIB.
ABS ---- AB
ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM,
         ED, AB, ST, CT, NA, RN, CN, GEN
BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED
DALL --- ALL, delimited for post processing
IABS --- ABS, with a text label
IALL --- ALL, indented with text labels
IBIB --- BIB, indented with text labels
IND ---- ST, CT, NA, RN, CN, GEN
SAM ---- TI, CM, ST, CT, NA, RN, CN, GEN
TRI ---- TI, CM, ST, CT, NA, RN, CN, GEN
TRIAL -- TI, CM, ST, CT, NA, RN, CN, GEN
HIT. ---- All fields containing hit terms
HITIND - IND
KWIC --- All hit terms plus 20 words on either side
OCC ---- List of display fields containing hit terms
Hit terms will be highlighted in all available fields except CM and
PY.
To display a particular field or fields, enter the display field
codes. For a list of display field codes, enter 'HELP DFIELDS'
at an arrow prompt (=>). Examples of formats include: 'BIB';
'AB'; 'SO,ST'. You may specify the format fields in any order,
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and the information will be displayed in the same order as the

11378 VINBLASTINE 10847 PACLITAXEL format specification.

The same formats (except for HIT, HITIND, KWIC, and OCC) may be used with the DISPLAY ACC command to display the record for a specified Accession Number.

ENTER DISPLAY FORMAT (BIB):d 1-2 bib abs
'D' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'
'1-2' IS NOT A VALID FORMAT FOR FILE 'MEDLINE'

The following are valid formats:

The default display format is BIB.

ABS ---- AB

ALL ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED, AB, ST, CT, NA, RN, CN, GEN

BIB ---- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED

CBIB --- AN, DN, TI, CM, AU, CS, NC, SO, CY, DT, LA, FS, OS, EM, ED

CBIB --- ALL, delimited for post processing

IABS --- ABS, with a text label

IALL --- ALL, indented with text labels

IBIB --- BIB, indented with text labels

IND ---- ST, CT, NA, RN, CN, GEN

SAM ---- TI, CM, ST, CT, NA, RN, CN, GEN

TRI ---- TI, CM, ST, CT, NA, RN, CN, GEN

TRIAL -- TI, CM, ST, CT, NA, RN, CN, GEN

HIT ---- All fields containing hit terms

HITIND - IND

KWIC --- All hit terms plus 20 words on either side

OCC ---- List of display fields containing hit terms

Hit terms will be highlighted in all available fields except CM and PY.

To display a particular field or fields, enter the display field codes. For a list of display field codes, enter 'HELP DFIELDS' at an arrow prompt (=>). Examples of formats include: 'BIB'; 'AB'; 'SO,ST'. You may specify the format fields in any order, and the information will be displayed in the same order as the format specification.

The same formats (except for HIT, HITIND, KWIC, and OCC) may be used with the DISPLAY ACC command to display the record for a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):end

=> s 15

1.6

500869 CANCER
12639 INTERPHASE
10 CYTODIERESIS
3491 CYTOKINESIS

2 L3 AND CANCER AND (INTERPHASE OR CYTODIERESIS OR CYTOKINESIS)

=> d 1-2 bib abs

L6 ANSWER 1 OF 2 MEDLINE on STN

AN 2003189192 MEDLINE

DN PubMed ID: 12708486

TI Usefulness of combined treatment with mild temperature hyperthermia and/or tirapazamine in the treatment of solid tumors: its independence of p53 status.

AU Masunaga Shin-ichiro; Ono Koji; Takahashi Akihisa; Ohnishi Ken; Ohnishi Takeo; Suzuki Minoru; Nagata Kenji; Kinashi Yuko; Nagasawa Hideko; Uto Yoshihiro; Hori Hitoshi

CS Radiation Oncology Research Laboratory, Research Reactor Institute, Kyoto

University, Noda, Kumatori-cho, Sennan-gun, Osaka 590-0494.. smasuna@rri.kyoto-u.ac.jp

- SO Cancer science, (2003 Jan) 94 (1) 125-33. Journal code: 101168776. ISSN: 1347-9032.
- CY Japan
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 200305
- ED Entered STN: 20030424

Last Updated on STN: 20030514

Entered Medline: 20030513

Human head and neck squamous cell carcinoma cells transfected with mutant AB TP53 (SAS/mp53) or with neo vector as a control (SAS/neo) were inoculated subcutaneously into both hind legs of Balb/cA nude mice. Mice bearing the tumors received 5-bromo-2'-deoxyuridine (BrdU) continuously to label all proliferating (P) cells in the tumors. The mice then received tirapazamine (TPZ) with or without mild temperature hyperthermia (40 degrees C, 60 min) (MTH), gamma-ray irradiation with or without MTH and/or TPZ, cisplatin (CDDP) with or without MTH and/or TPZ, or paclitaxel (TXL) with or without MTH and/or TPZ. After each treatment, the tumors were excised, minced and trypsinized. The tumor cell suspensions thus obtained were incubated with a cytokinesis blocker (cytochalasin-B), and the micronucleus (MN) frequency in cells without BrdU labeling (i.e., quiescent (Q) cells) was determined by using immunofluorescence staining for BrdU. Meanwhile, 6 h after gamma-ray irradiation or 24 h after other cytotoxic treatments, tumor cell. suspensions obtained in the same manner were used for determining the frequency of apoptosis in Q cells. The MN frequency and apoptosis frequency in total (P+Q) tumor cells were determined from the tumors that were not pretreated with BrdU. On the whole, gamma-ray irradiation and CDDP injection induced a higher frequency of apoptosis and lower frequency of MN in SAS/neo cells than SAS/mp53 cells. There were no apparent differences in the induced frequency of apoptosis and MN between SAS/neo and SAS/mp53 cells after TPZ or TXL treatment. MTH sensitized cells to TPZ-inducing cytotoxicity more markedly in SAS/mp53 and Q cells than in SAS/neo cells and total cells, respectively. In gamma-ray irradiation and CDDP treatment, the enhancement in combination with MTH and/or TPZ was more remarkable in SAS/mp53 cells and Q cells than in SAS/neo and total tumor cells, respectively. Also in the case of TXL treatment, the combination with MTH and/or TPZ induced a slightly greater enhancement effect in SAS/mp53 cells and Q cells. In view of the difficulty in controlling mutated p53 status tumors and intratumor Q cells, combination treatment with MTH and/or TPZ as a cooperative modality in cancer therapy is considered to have potential for controlling solid tumors as a whole.

- L6 ANSWER 2 OF 2 MEDLINE on STN
- AN 97206774 MEDLINE
- DN PubMed ID: 9102841
- TI [Significance of formation of micronuclei in SCC VII murine cells treated with various chemotherapeutic agents].

 Znacaj formiranja mikronukleusa u murinim celijama SCC VII tretiranih razlicitim hemioterapijskim agensima.
- AU Jeremic B; Sibamoto J; Abe M
- CS Department of Oncology, Clinical-Hospital Centre, Kragujevac, Yugoslavia.
- SO Srpski arhiv za celokupno lekarstvo, (1996 Jul-Aug) 124 (7-8) 169-74. Journal code: 0027440. ISSN: 0370-8179.
- CY Yugoslavia
- DT Journal; Article; (JOURNAL ARTICLE)
- LA Serbian
- FS Priority Journals
- EM 199704
- ED Entered STN: 19970424

Last Updated on STN: 19970424

Entered Medline: 19970416

AB

INTRODUCTION: The in vitro chemosensitivity testing aims at predicting the response of an individual tumour to chemotherapy choosing optimal agents for a particular patient. Among many chemosensitivity tests developed over the years [1-6], special emphasis was made on clonogenic assays that showed good use and correlation between laboratory and clinical data [7-9]. One of the assays used to predict the response to various anticancer modalities is the micronucleus assay using the cytokinesis-block [12-14]. This block is achieved by administration of Cytochalasin-B in order to prevent cytoplasmic, but not the nuclear, division. This leads to micronucleus formation which are counted in binuclear cells. Since there are only a few reports of the use of this assay in predicting chemosensitivity [13, 16], we explored the possibility of using this assay to predict chemosensitivity to various anti-cancer agents. MATERIAL AND METHODS: Exponentially growing SCC VII cells were treated with various concentrations of 11 anti-cancer agents: Mitomycin C, Doxorubicin (ADR), Epirubicin (EPI), Cisplatin, Carboplatin (CBDCA), Etoposide (VP-16), Vincristine, 5-fluorouracil, Methotrexate, Nimustine, and Dacarbazine for 1 hour. After that, Cytochalasin-B was added and dishes were incubated. After various time intervals, cells were fixed in situ and dried. Electron microscope was used to count the number of micronuclei (MN) in binucleate cells as . well as multinucleate cells (MNC) in the total cell population. Cell survival was also evaluated by using the colony formation assay [18]. RESULTS: Maximal % of binucleate cells (BNC) was usually reached at 24-30 hours of culture, except for cells treated with ADR and EPI, in which it was reached at 30-72 hours (Figures 1 and 2). All drugs induced formation of micronuclei and dose-response curves for micronucleus frequency were obtained using the data at peak % BNC times. For all drugs, micronucleus frequency increased with concentration (Figure 3), but at the highest concentration used (considered to be overly toxic-Figure 4), the micronucleus frequency was rather lower. This decrease in micronucleus frequency was largely attributed to the decrease in % BNC. When the data at the highest concentrations of all drugs were excluded, a correlation was found between micronucleus frequency and surviving fraction (r = 0.85; p < 0,001) (Figure 5). DISCUSSION: Since micronucleus formation is a sign of chromosome damage that leads to cell death, we used this assay to evaluate chemosensitivity in 11 widely used anticancer agents. Although they can be classified according to mechanism of action as different class agents, they have in common the formation of micronuclei as a sign of cytotoxicity. Cell cycle arrest observed in some agents might be evaluated by assessing the delay in increase of BNC and MNC. The difference observed regarding cell cycle arrest suggested different mechanisms of its action. MN frequency was almost dose-dependent at lower concentrations, but at the highest concentrations, it obviously decreased, showing, therefore, some discrepancies with the data obtained when radiosensitivity was tested that way [14], probably due to extreme toxicity of agents. The optimal concentrations seem to be those providing a 20-80% surviving fraction. Another slight difference, when compared with similar radiosensitivity studies is a decrease with longer duration of culture observed in chemosensitivity testings. The reason for this difference is still unknown, but it emphasized the necessity for choosing the optimal duration of culture, probably that necessary for reaching maximal % BNC. This assay seems useful in predicting chemosensitivity of at least some tumour cells to various (appropriate) concentrations of various anti-cancer agents. However, new studies are warranted to further use of this assay, before testing it in clinical practice.

=> d 20-29 bib abs

- L7 ANSWER 20 OF 29 MEDLINE on STN
- AN 91369816 MEDLINE
- DN PubMed ID: 1892755
- TI Resistance to the antimitotic drug estramustine is distinct from the multidrug resistant phenotype.
- AU Speicher L A; Sheridan V R; Godwin A K; Tew K D
- CS Department of Pharmacology, Fox Chase Cancer Center, Philadelphia, Pennsylvania 19111.
- SO British journal of cancer, (1991 Aug) 64 (2) 267-73. Journal code: 0370635. ISSN: 0007-0920.
- CY ENGLAND: United Kingdom
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 199110
- ED Entered STN: 19911108

Last Updated on STN: 19970203 Entered Medline: 19911022

Following EMS mutagenesis, three estramustine (EM) resistant DU 145 human AB prostatic carcinoma cell lines were clonally selected by exposure to incrementally increasing concentrations of the drug. Although only low levels of resistance (approximately 3-fold) were attainable, this resistance was stable in the absence of continuous drug exposure. These EM-resistant clones (EMR 4,9,12) did not exhibit cross resistance to vinblastine, taxol, or adriamycin, and had collateral sensitivity to cytochalasin B. None of the lines had elevated expression of P-glycoprotein mRNA or glutathione S-transferase activity, suggesting a phenotype distinct from the classic multi-drug resistance phenotype. conclusion was supported further by the observation that two MDR cell lines (FLC mouse erythroleukaemic and SKOV3 human ovarian carcinoma cells) showed sensitivity to EM. Fluorescent activated cell sorting analysis of the effects of EM on cell cycle traverse revealed that at EM concentrations up to 20 microM an increasing percentage of wild type cells were blocked in G2/M; no such effect occurred in EMR lines. Differential interference contrast microscopy was employed to study EM's effect on mitosis. EMR lines were able to form functional, albeit smaller, spindles at EM concentrations that resulted in chromosomal disorganisation and inhibition of mitotic progression in wild type cells. EMR lines were able to progress through mitosis and cytokinesis at the same rate as untreated cells. Tritiated EM was used to evaluate potential drug uptake/efflux mutations in ERM clones. EMR 4 and 9 incorporate less EM than wild type cells; however, they have significantly decreased cellular volumes. The initial efflux rate constants for EMR clones were greater than for wild type cells. Within 5 min greater than 70% of the drug was lost from resistant cells compared to a 50% loss by the wild type. Although the specific mechanisms of resistance have yet to be defined, the lack of collateral resistance to other MDR/anti-microtubule agents could serve as the basis for the clinical use of EM in combination chemotherapy.

- L7 ANSWER 21 OF 29 MEDLINE on STN
- AN 91029202 MEDLINE
- DN PubMed ID: 2121336
- TI Cytotoxic effects of **cell cycle** phase specific agents: result of **cell cycle** perturbation.
- AU Kung A L; Zetterberg A; Sherwood S W; Schimke R T
- CS Department of Biological Sciences, Stanford University, California 94305.
- NC GM-14931 (NIGMS)
- SO Cancer research, (1990 Nov 15) 50 (22) 7307-17.
 Journal code: 2984705R. ISSN: 0008-5472.
- CY United States

```
Journal; Article; (JOURNAL ARTICLE)
DT
LA
     English
     Priority Journals
FS
EM
     199012
     Entered STN: 19910208
ED
     Last Updated on STN: 19910208
     Entered Medline: 19901207
     Although agents which act in a cell cycle phase
AΒ
     specific manner are commonly used in the clinic and in basic research, it
     is as yet unclear why these agents are cytotoxic. In this paper, we
     examine the cellular events associated with the cytotoxicity of
     aphidicolin and vincristine in CHO strain AA8 cells. Cell
     killing resulting from aphidicolin treatment was found to require a period.
     of inhibition-free growth following removal of the drug and was associated
     with characteristic aberrant mitotic processes. The cytotoxic effects of
     aphidicolin could be antagonized by the concomitant inhibition of protein
     synthesis with cycloheximide in the period of DNA synthesis inhibition.
     Cell killing resulting from treatment with vincristine was
     associated with the aberrant segregation of nuclear material and the
     formation of multiple partial nuclei. Vincristine cytotoxicity
     was found to be antagonized by concomitant administration of cycloheximide
     or cytochalasin D. These data support a hypothesis that the
     cytotoxic effects of cell cycle phase specific agents
     do not derive directly from their biochemical actions per se. We propose
     that cell death results from processes that are evoked by dissociation of
     normally integrated cell cycle events, and that
     dissociation involves replicative/mitotic events in the case of
     aphidicolin and karyokinetic/nuclear reformation events in the case of
     vincristine.
                         MEDLINE on STN
L7
     ANSWER 22 OF 29
     87301802
                  MEDLINE
AN
     PubMed ID: 2887300
DN
     Effects of cytoskeletal inhibitors on water proton relaxation time changes
ΤI
     in unfertilized and fertilized sea urchin eggs.
     Zimmerman S; Zimmerman A M; Cameron I L; Fullerton G D; Schatten H;
ΑU
     Schatten G
     HD-17087 (NICHD)
NC
     HD12913 (NICHD)
     HD22902 (NICHD)
     Cell biology international reports, (1987 Aug) 11 (8) 605-14.
SO
     Journal code: 7708050. ISSN: 0309-1651.
CY
     ENGLAND: United Kingdom
DT
     Journal; Article; (JOURNAL ARTICLE)
     English
LA
FS
     Priority Journals; Space Life Sciences
EM
     198710
ED
     Entered STN: 19900305
     Last Updated on STN: 19970203
     Entered Medline: 19871020
     Unfertilized and fertilized sea urchin eggs were used for pulsed proton
AB
     NMR spin-lattice relaxation time (T1) measurements of cellular water. An
     81% increase in T1 time at fertilization was largely explained by the
     accumulation of extracellular water in the perivitelline space. To assess
     the role of microtubule and actin filament assembly and disassembly, eggs
     were treated with drugs that are known to change these cytoskeletal
     elements (i.e., colchicine, taxol and cytochalasin B). Egg
     volume was also monitored in all studies to rule out the influence of
     water content changes on the observed T1 relaxation time changes. Neither
     assembly nor disassembly of microtubules changed the T1 relaxation time.
     The role of actin polymerization and depolymerization is discussed as a
```

possible explanation for the observed cell cycle dependent water proton T1 relaxation time changes.

```
ANSWER 23 OF 29
                        MEDLINE on STN
L7
                 MEDLINE
AN
     83004003
     PubMed ID: 6126383
DN
     The effects of cyclosporins on the cell cycle of
TI
     t-lymphoid cell lines.
     Koponen M; Grieder A; Loor F
ΑU
     Experimental cell research, (1982 Aug) 140 (2) 237-50.
SO
     Journal code: 0373226. ISSN: 0014-4827.
CY
     United States
DT
     Journal; Article; (JOURNAL ARTICLE)
LA
     English
FS
     Priority Journals
EM
     198212
ED
     Entered STN: 19900317
     Last Updated on STN: 19950206
     Entered Medline: 19821203
L7
     ANSWER 24 OF 29
                         MEDLINE on STN
                  MEDLINE
AN
     81264473
     PubMed ID: 7263771
DN
     Effects of cytoskeletal disrupting agents on replication of bovine
ΑU
     Selden S C 3rd; Rabinovitch P S; Schwartz S M
     AG-01751 (NIA)
NC
     HL-03174 (NHLBI)
     HL-18645 (NHLBI)
    Journal of cellular physiology, (1981 Aug) 108 (2) 195-211.
     Journal code: 0050222. ISSN: 0021-9541.
CY
     United States
     Journal; Article; (JOURNAL ARTICLE)
DT
LA
     English
     Priority Journals ·
FS
ΕM
     198110
     Entered STN: 19900316
     Last Updated on STN: 19970203
     Entered Medline: 19811025
     Colchicine and vinblastine inhibited endothelial cell migration
AB
     but had no effect on the stimulation of replication seen at wound edges in
     cultures of endothelium at stationary density. This is in contrast to the
     effects of cytochalasins which inhibit both migration and replication at
     wound edges. Moreover, colchicine and vinblastine stimulated
     cell replication in the unwounded, confluent monolayer. This effect has
     kinetics similar to the stimulation of replication at a wound edge and is
     associated with an initial retraction of cell borders, leaving gaps
     between cells. Cytochalasin D inhibited the growth response to
     microtubule disrupting agents but did not prevent cell retraction.
     Stimulation of replication by microtubule disrupting agents was not
     dependent on serum but was synergistic with serum in cultures rinsed
     repeatedly with serum-free medium. The replication occurred prior to any
     cell loss. When, however, cells were allowed to complete mitosis, about
     one-half of the daughter cells detached from the monolayer so that there
     was no increase in cell density. We conclude that microtubule disrupting
     agents are the first agents found to be effective in stimulating growth of
     vascular endothelium at saturation density. These data further suggest
     that colchicine and vinblastine stimulate cell growth in a
     manner similar to wounding, where cell movement is a prerequisite to cell
     replication.
L7
     ANSWER 25 OF 29
                         MEDLINE on STN
     80067295
                  MEDLINE
AN
DΝ
     PubMed ID: 159787
     The cytoplasmic origin of variability in the timing of S phase in
     mammalian cells.
```

Cell biology international reports, (1979 Dec) 3 (9) 707-16.

ΑU

SO

Brooks R F

Journal code: 7708050. ISSN: 0309-1651.

- CY ENGLAND: United Kingdom
- Journal; Article; (JOURNAL ARTICLE) DT
- LA English
- FS Priority Journals
- EM 198002
- ED Entered STN: 19900315

Last Updated on STN: 19970203 Entered Medline: 19800226

- The time at which S phase begins in mammalian cells is highly variable AΒ with respect to cell age. Evidence is presented that this variability does not arise because the initiation of DNA synthesis depends on the stochastic interaction of an initiator substance with a rare initiation site. Instead, the signal responsible for starting S phase must appear at random in the cytoplasm and may be transient.
- ANSWER 26 OF 29 MEDLINE on STN L7
- AN 79180175 MEDLINE
- DN PubMed ID: 286310
- Production of a tissue-like structure by contraction of collagen lattices TI by human fibroblasts of different proliferative potential in vitro.
- Bell E; Ivarsson B; Merrill C AU
- SO Proceedings of the National Academy of Sciences of the United States of America, (1979 Mar) 76 (3) 1274-8. Journal code: 7505876. ISSN: 0027-8424.
- CY United States
- Journal; Article; (JOURNAL ARTICLE) DT
- LA English
- FS Priority Journals
- ΞM 197907
- Entered STN: 19900315 ΞD Last Updated on STN: 19970203 Entered Medline: 19790725
- AB Fibroblasts can condense a hydrated collagen lattice to a tissue-like structure 1/28th the area of the starting gel in 24 hr. The rate of the process can be regulated by varying the protein content of the lattice, the cell number, or the concentration of an inhibitor such as Colcemid. Fibroblasts of high population doubling level propagated in vitro, which have left the cell cycle, can carry out the contraction at least as efficiently as cycling cells. The potential uses of the system as an immunologically tolerated "tissue" for wound healing and as a model for studying fibroblast function are discussed.
- ANSWER 27 OF 29 MEDLINE on STN L7
- AN 78082280 MEDLINE
- PubMed ID: 620423 DN
- Inhibition of mycoplasma cell division by cytochalasin B. ΤI
- Ghosh A; Maniloff J; Gerling D A Cell, (1978 Jan) 13 (1) 57-64. AU
- SO

Journal code: 0413066. ISSN: 0092-3674.

- CY ENGLAND: United Kingdom
- DTJournal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 197803
- Entered STN: 19900314 Last Updated on STN: 19900314

Entered Medline: 19780321

Mycoplasma gallisepticum has subcellular organelles which may function as a primitive "mitotic-like" apparatus. To investigate these further, we have studied the effects of cytochalasin B (CB) on M. gallisepticum. We found that CB inhibits cell division; this is the only procaryote thus far reported to be inhibited by CB. CB does not inhibit glucose or macromolecule precursor uptake. It stops cellular DNA synthesis, however, although RNA and protein synthesis continue (at a

reduced rate). CB removal results in a resumption of DNA synthesis, followed by cell division. There appears to be some degree of cell synchrony in this first division after CB removal. These results, together with morphological data, indicate that CB blocks at two points in the cell cycle: at the time "mitotic-like" structures are formed and at the time of cell division. It is suggested that the CB blocks may result from a disruption of actin-like protein structures required at these points in the cell cycle.

- ANSWER 28 OF 29 MEDLINE on STN L7
- AN 77114120 MEDLINE
- DN PubMed ID: 189934
- Decreased adherence to the substrate in Rous sarcoma virus-transformed TI chicken embryo fibroblasts.
- ΑU
- Weber M J; Hale A H; Losasso L Cell, (1977 Jan) 10 (1) 45-51. SO

Journal code: 0413066. ISSN: 0092-8674.

- CY ENGLAND: United Kingdom
- Journal; Article; (JOURNAL ARTICLE) DT
- English LA
- Priority Journals FS
- 197704 EΜ
- Entered STN: 19900313

Last Updated on STN: 19970203

Entered Medline: 19770425

- Cell-substrate adherence in cultures of chicken embryo fibroblasts was AB examined by determining the number of cells which could be detached from the culture dish by a stream of medium. Transformed cells were significantly less adherent than their normal counterparts. In cultures infected with a mutant of Rous sarcoma virus which is temperatureconditional for transformation, adherence changed promptly following a temperature shift. This change did not require progression through the cell cycle. The transformation-specific decrease in adherence required new protein synthesis, but the restoration of adherence which occurred following a shift to the restrictive temperature could occur in the absence of new protein synthesis. Inhibitor experiments suggested the importance of microfilaments and perhaps microtubules in the changes in detachability. In addition, there was a positive correlation between levels of surface LETS protein and cell substrate adherence following a temperature shift, although it seems probable that the bulk of the surface LETS is neither necessary nor sufficient for maintenance of normal cell substrate adherence.
- L7 ANSWER 29 OF 29 MEDLINE on STN
- AN 77106339 MEDLINE
- DN PubMed ID: 65050
- TI Cell cycle-dependent inhibition of Kirsten Murine sarcoma-leukemia virus release by cytochalasin B.
- ΑU
- SO Virology, (1977 Jan) 76 (1) 146-51. Journal code: 0110674. ISSN: 0042-6822.
- CY United States
- DTJournal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EΜ 197703

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ED Entered STN: 19900313

Last Updated on STN: 19970203

Entered Medline: 19770315

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS SINCE FILE TOTAL

FULL ESTIMATED COST ENTRY SESSION 12.80 15.37

STN INTERNATIONAL LOGOFF AT 18:55:09 ON 29 JUN 2005